REMARKS/ARGUMENTS

The Claims are 1-4, 7-9 and 12-13. Claim 1 has been amended. Claims 5-6, 9-11 and 14-20 are cancelled without prejudice or disclaimer. No new subject matter has been added.

Claims 1-4, 7 and 12-13 stand rejected under 35 U.S.C. § 102(b) as being allegedly anticipated by Neubauer et al., Virology, 1997, Vol. 239, pp. 36-45 ("Neubauer"). The Examiner has alleged that Neubauer discloses an EHV-1 mutant devoid of the open reading frame of gM. Applicants respectfully disagree with this description of the teachings of Neubauer.

Neubauer discloses a modified EHV-1 isolate (HDgM), which includes an insertion within the gene 52, which encodes glycoprotein M (gM). Construction of the HDgM isolate is described in Osterrieder et al., 1996 (J. Virology, Vol. 70, pages 4110-4115). A summary is also found in Osterrieder et al., 2001 (Vet. Microbiology, Vol. 81, pages 219-226), which also summarizes the results and benefits of the claimed invention. The HDgM described in Neubauer was constructed by an insertion of the E.coli lacZ gene within the open reading frame of the gene 52 (gM). Thus, the HDgM encodes for the whole gene 52. In contrast, the claimed invention is directed to an EHV, wherein the nucleotide sequence encoding a protein gM is at least 80% absent. The inventive EHV is construed by a replacement of the Sph I - Hinc II polynucleotide fragment, encoding for about 75% of gM protein (see Osterrieder et al., 2001, page 221, figure 1 and page 219, abstract, line 3).

Moreover, Neubauer does not disclose or teach about an EHV-1 mutant devoid of the open reading frame of gM. Neubauer alleged to have provided an EHV gM mutant, which does not express a gM protein or part thereof. However, it was shown in the present patent application (page 8, lines 1-9 and Figure 3b, lane 2) and also by one of the co-authors of Neubauer (Klaus Osterrieder in Osterrieder et al., 2001, page 220, last sentence to page 222, line 14, and figure 2, right image lane 2), that the EHV HDgM disclosed in Neubauer expressed a truncated gM-protein of 28 kDa, that demonstrate an immunomodulatory potential similar to the gM-producing parent strain. The appearance of the 28kDa is also in line with the teaching of Osterrieder et al., 1996 (Virology, Vol.

232, pages 230-239), who assumed that a 28 kDa gM protein results from a translation start at methionine 197 of the 450 amino acid full lengths gM protein (Osterrieder et al., 1996, page 234, right column, last sentence). Thus, contrary to the allegation of the authors of Neubauer, the insertion of the E.coli lacZ gene within the gM protein, does not result in an EHV-mutant, wherein at least 80% of the nucleotide sequence of gM protein is absent, nor in an EHV-mutant that does not express a gM protein or a part thereof. Thus, Neubauer does not describe or teach an EHV negative gM mutant.

Accordingly, Neubauer does not anticipate the present invention, and Applicants respectfully request the withdrawal of this rejection.

Claims 1-4, 7-8 and 12-13 also stand rejected under 35 U.S.C. § 103(a) as allegedly obvious over Neubauer. The Examiner has alleged that it would be obvious to the ordinary artisan to assemble the components disclosed in Neubauer into a kit as a convenient means of using the disclosed pharmaceutical composition as a protective vaccine. The motivation being the disclosure that mice infected with L11ΔgM were protected against challenge with RacL11 virus.

However, as stated above, Neubauer does not disclose or suggest an EHV negative gM mutant, since its purported teachings are incorrect. Thus, a skilled artisan could not possibly arrive at the present invention from the teaching of Neubauer because it is non-enabling. A prior art reference must enable the making of the claimed invention in order to support a rejection based upon obviousness. See In re Hoeksemer, 399 F.2d 269, 158 USPQ 596 (CCPA 1968). See also MPEP § 2144.08 (II) (B). Additionally, one of the three requirements for a prima facia case of obviousness is that the prior art teach or suggest all the limitations of the clinical invention. See MPEP § 2143. Neubauer does not teach or suggest an EHV-mutant, wherein at least 80% of the nucleotide sequence of gM protein is absent.

Finally, it has been surprisingly found, that the claimed EHV isolates, wherein the nucleotide sequence encoding a protein gM is at least 80% absent and wherein the expression of the gene coding for the UL9 homolog (gene 53) is not affected, when used as a vaccine, resulted in a better performance of the vaccinated animals than any prior art EHV or EHV mutant. In contrast to the wild type EHV and to that described in Neubauer,

the claimed EHV mutants resulted in an enhanced and unpredictable immunogenic property. Animals vaccinated with the EHV gM mutants according to the present invention resulted in a higher mean body weight and a reduced loss of weight gain after challenge with an infectious wild type virus (see Example 1 of the current patent application). None of the prior art documents teach or suggest that gM negative EHV mutants would result in such an effect.

For the above stated reasons, Applicants respectfully suggest that the present invention cannot be obvious in view of Neubauer and respectfully request withdrawal of this rejection.

In view of the foregoing, it is respectfully submitted that the subject application is in condition for allowance and such favorable action at an early date is earnestly solicited.

Respectfully submitted,

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